

P36**Monitoring of procalcitonin, IL-6 and brain natriuretic peptide for sepsis diagnosis in cardiac surgery****R Barchetta, C Alessandrini, C Di Corato, F Candidi, F Turani, M Falco***Department of Anaesthesia and Intensive Care, European Hospital, Rome, Italy**Critical Care 2009, 13(Suppl 4):P36 (doi: 10.1186/cc8092)*

Introduction Procalcitonin (PCT) and IL-6 are markers used in the evaluation of systemic inflammation (SIRS) and septic states. The purpose of this study is to analyse changes in plasma concentrations of PCT and IL-6 in patients undergoing cardiac surgery on-pump and assess its reliability in the early detection of post-operative infectious complications. In all patients the variation of brain natriuretic peptide (BNP) was also evaluated in order to stratify the clinical condition of patients.

Methods We measured serum levels of PCT, IL-6 and BNP in adult patients undergoing myocardial revascularization and/or valve surgery performed in extracorporeal circulation. The measurements were performed on the day before the intervention (T0), at the end of surgery (T1) and then until the third and fourth postoperative day (T2 to T4). We also recorded the onset of cardiac, respiratory, neurological, renal and septic complications. The diagnosis of sepsis was confirmed retrospectively on the basis of clinical, radiological and microbiological data. All data are expressed as mean and standard deviation. The Kruskal-Wallis test was used to assess changes over time of variables. $P < 0.05$ was considered statistically significant.

Results There have been enrolled 60 patients undergoing cardiac surgery in extracorporeal circulation. Among these, nine patients developed septic complications. The results of temporal changes and the significance are presented in Table 1.

Table 1 (abstract P36)**Results of temporal changes and significance**

	T0	T1	T2	T3	T4
PCT (ng/ml)					
Nonseptic	0.04	0.04	0.58	0.34	0.34
Septic	0.04	0.15	2.63	1.87	0.74
P	NS	<0.001	<0.001	<0.001	<0.01
IL-6 (pg/ml)					
Nonseptic	12	160	129	78	75
Septic	18	184	145	261	92
P	NS	NS	NS	<0.01	NS
BNP					
Nonseptic	159	154	347	428	492
Septic	373	627	731	756	798
P	<0.01	<0.01	<0.01	<0.05	<0.05

Conclusions In patients who develop septic complications, changes in PCT occur earlier than changes in IL-6. Furthermore, BNP performs in the same fashion as PCT and correlates better than IL-6 with the clinical data of the infection status. In conclusion, monitoring PCT seems to be useful in early diagnosis of septic complications in patients undergoing cardiac surgery and more sensitive on the variations in IL-6. The combined study of variations in PCT and BNP could improve the diagnostic accuracy in these patients.

P37**T-cell-specific peroxisome proliferator-activated receptor gamma depletion inhibits T-cell apoptosis and improves survival of septic mice via an IL-2-dependent mechanism****MV Schmidt¹, P Paulus², A-M Kuhn¹, V Meilladec-Jullig¹, K Zacharowski², B Bruene¹, A von Knethen¹***¹Institute of Biochemistry I, Faculty of Medicine, Goethe-University Frankfurt, Germany; ²Department of Anaesthesia, Intensive Care Medicine & Pain Therapy, University Hospital Frankfurt, Germany**Critical Care 2009, 13(Suppl 4):P37 (doi: 10.1186/cc8093)*

Introduction Immune paralysis with massive T-cell apoptosis is a central pathogenic event during sepsis and correlates with septic patient mortality. Previous observations implied a crucial role of peroxisome proliferator-activated receptor gamma (PPAR γ) during T-cell apoptosis.

Methods To elucidate mechanisms of PPAR γ -induced T-cell depletion, we used an endotoxin model as well as the caecal ligation and puncture sepsis model to imitate septic conditions in wild-type versus conditional PPAR γ knockout (KO) mice.

Results PPAR γ KO mice showed a marked survival advantage compared with control mice. Their T cells were substantially protected against sepsis-induced death and showed a significantly higher expression of the pro-survival factor IL-2. Since PPAR γ is described to repress nuclear factor of activated T cells (NFAT) transactivation and concomitant IL-2 expression, we propose inhibition of NFAT as the underlying mechanism allowing T-cell apoptosis. Corroborating our hypothesis, we observed up-regulation of the pro-apoptotic protein BIM and downregulation of the anti-apoptotic protein Bcl-2 in control mice, which are downstream effector proteins of IL-2 receptor signaling. Application of a neutralizing anti-IL-2 antibody reversed the pro-survival effect of PPAR γ -deficient T cells and confirmed IL-2-dependent apoptosis during sepsis.

Conclusions Apparently antagonizing PPAR γ in T cells might improve their survival during sepsis, which concomitantly enhances defence mechanisms and possibly provokes an increased survival of septic patients.

P38**Induction of severe *Staphylococcus aureus* sepsis in pigs****TM Iburg¹, PS Leifsson¹, M Kjelgaard-Hansen², P Heegaard³, B Wiinberg², B Aalbaek¹, AE Olsson¹, MGS Hansen¹, LB Thomsen¹, HE Jensen¹, JS Agerholm¹, OL Nielsen¹***¹Department of Veterinary Disease Biology, and ²Department of Small Animal Clinical Sciences, Faculty of Life Sciences, University of Copenhagen, Denmark; ³Department of Veterinary Diagnostics and Research, National Veterinary Institute, Technical University of Denmark, Copenhagen, Denmark**Critical Care 2009, 13(Suppl 4):P38 (doi: 10.1186/cc8094)*

Introduction Organ dysfunction is an integrated part of severe sepsis, and severe sepsis is one of the major causes of death in ICUs. Lately Gram-positive bacteria accounted for more than one-half of the overall sepsis cases reported in the USA, with *Staphylococcus aureus* being the most commonly isolated bacterium. Effective treatment of sepsis is still not optimal and good animal models are needed for research in pathogenesis and treatment. *S. aureus* infections are also common in pigs and are isolated from approximately 40% of embolic lesions found in slaughter-pigs.

Objective To establish a porcine model of severe sepsis.